Section: Biochemistry

Original Research Article

STATUS OF VITAMIN D LEVELS IN HYPOTHYROID PATIENTS AND ITS ASSOCIATION WITH THYROID PROFILE IN SUBGROUP OF POPULATION OF SOUTH EAST DELHI

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ABSTRACT

Background: Thyroid hormones and vitamin D both function through steroid receptors, and because their response elements on genes are similar, they may influence one another's actions. Limited studies are available on this subject and the exact relationship between these parameters in predicting vitamin D association with hypothyroidism is not well established.

Materials and Methods: In this study, diagnosed cases of 90 hypothyroid patients were taken from HAHC Hospital and categorized into subclinical hypothyroid (n = 45) and overt hypothyroid (n = 45). TSH, FT3, and FT4 were analyzed by an automated analyzer based on CMIA (Chemiluminescent Microparticle Immunoassay). Additionally, 90 euthyroid subjects were taken as controls.

Results: Our study revealed the presence of vitamin D deficiency in both subclinical and overt hypothyroid patients as compared to euthyroid subjects. Pearson correlation analysis on individual patient data (n = 90 hypothyroid subjects) demonstrated a statistically significant negative correlation of vitamin D with TSH and significant positive correlations with T3 and T4.

Conclusion: Our study shows that vitamin D deficiency is more pronounced in hypothyroid patients and correlates with poorer thyroid function, supporting routine screening and supplementation to enable timely intervention.

Keywords: Subclinical hypothyroidism, Overt hypothyroidism, Chemiluminescent Microparticle Immunoassay, Vitamin D.

INTRODUCTION

Vitamin D is a fat-soluble vitamin crucial for calcium metabolism and bone health. In recent years, it has also been demonstrated to have a number of effects on extraskeletal health, including a bearing on cellular differentiation, maturation, proliferation, apoptosis, angiogenesis, and cell growth.[1] Circulating vitamin D exists primarily in two metabolic forms: 25-hydroxyvitamin D [$25(OH)D_3$] and the biologically active 1,25-dihydroxyvitamin D [$1,25(OH)_2$ D_3]. Since $25(OH)D_3$ has a relatively long circulatory half-life of approximately 15 days and reflects the body's overall vitamin D stores, it is the preferred marker in both clinical practice and research.[2] Despite living in one of the sunniest countries in the world, India paradoxically reports some of the highest rates of vitamin D deficiency globally, affecting people irrespective of age, income level, or whether they live in a city or village. Insufficient vitamin D status has been implicated as a contributing factor in a range of chronic and infectious conditions prevalent in India, from skeletal disorders such as rickets and osteoporosis to cardiovascular disease, diabetes mellitus, several malignancies, and tuberculosis.[3]

Thyroid disorders tell a similarly concerning story. They remain the most prevalent endocrine conditions

in India, affecting an estimated 42 million people worldwide.[2,4,5] In Delhi alone, the combined prevalence of subclinical and overt hypothyroidism has been reported at 13.2%, with inland cities recording consistently higher rates than coastal regions, a disparity that points to both dietary and environmental contributors.[5] What makes this situation more complex, and arguably more interesting, is the growing body of evidence suggesting that vitamin D deficiency and hypothyroidism do not simply coexist by coincidence. Both thyroid hormones and vitamin D signal through nuclear steroid hormone receptors, and their respective gene response elements share striking structural similarity, raising the possibility that these two systems actively influence one another.[2] The vitamin D receptor (VDR), widely expressed across human tissues including the thyroid gland and pituitary, mediates the genomic actions of $1,25(OH)_2 D_3$. Disruption of VDR-mediated signalling has been shown to impair immune tolerance, potentially contributing to the autoimmune destruction of thyroid tissue that underlies a significant proportion of hypothyroidism cases.[2,4-11] Adding another layer to this picture, over 470 single nucleotide polymorphisms in the VDR gene have been identified, with Fok1 and Apa1 among the most functionally significant, influencing how individuals respond immunologically to vitamin D.[4] A reduced vitamin D level is therefore likely to worsen the systemic irregularities already linked to hypothyroidism.[2] While studies from north India have begun to explore the relationship between vitamin D and thyroid function, data from specific urban subpopulations, particularly south-east Delhi, remain conspicuously scarce. No major study has been conducted in this demographic to date, leaving a meaningful gap in our understanding of how these two conditions interact at a local, population-specific level. The present study was therefore undertaken to address this gap: to evaluate serum vitamin D levels in patients with subclinical and overt hypothyroidism, and to analyse their association with thyroid hormone profiles in a subgroup of the population of south-east Delhi with the hope that the findings may eventually inform more targeted, region-specific clinical practice.

MATERIALS AND METHODS

This was a hospital-based cross-sectional study conducted in the Department of Biochemistry, Hamdard Institute of Medical Sciences and Research (HIMSR) and HAHC Hospital, Jamia Hamdard, New Delhi, over a period of eight months from August 2023 to March 2024. Ethical approval was obtained from the Institutional Ethics Committee prior to commencement of the study (Approval No. HIMSR/IEC/00161/2023), and the study was conducted in accordance with the Declaration of Helsinki (2013 revision). Written informed consent was obtained from all participants prior to enrolment.

The study comprised 90 treatment-naïve adults with biochemically confirmed hypothyroidism attending the Medicine Outpatient Department (OPD) of HAHC Hospital, and 90 euthyroid adults recruited as controls. Patients were categorised into two subgroups: subclinical hypothyroidism (n = 45) and overt hypothyroidism (n = 45).

Inclusion Criteria

Adult patients (aged ≥ 18 years) with biochemically confirmed hypothyroidism were included. Subclinical hypothyroidism was defined as serum TSH > 4.94 µIU/mL with normal serum FT3 (1.88–3.18 pg/mL) and normal serum FT4 (0.70–1.48 ng/dL). Overt hypothyroidism was defined as serum TSH > 7 µIU/mL with low serum FT3 (< 1.88 pg/mL) and low serum FT4 (< 0.70 ng/dL). Only patients who were treatment-naïve (not on any thyroid hormone replacement therapy at the time of recruitment) and who provided written informed consent were enrolled.

Exclusion Criteria

The following were excluded from the study: pregnant women; individuals with a history of chronic alcoholism; patients with other thyroid disorders (hyperthyroidism, thyroiditis, or thyroid malignancy); patients currently receiving vitamin D supplementation or calcium supplementation; and individuals with known hepatic disease, renal disease, or malabsorption syndromes, as these conditions may independently affect vitamin D metabolism.

Biochemical Analysis: Venous blood samples were collected under fasting conditions in the morning to minimise diurnal variation. Serum was separated by centrifugation and analysed on the same day. Serum levels of thyroid-stimulating hormone (TSH), free triiodothyronine (FT3), and free thyroxine (FT4), along with serum 25-hydroxyvitamin D [$25(OH)D_3$], were all measured using the Abbott ARCHITECT i1000SR automated analyser based on Chemiluminescent Microparticle Immunoassay (CMIA). Vitamin D status was classified according to standard thresholds: deficiency (< 20 ng/mL), insufficiency (20–29 ng/mL), and sufficiency (≥ 30 ng/mL).

Statistical Analysis: All data were analysed using SPSS version 16.0 for Windows (IBM Corp., USA). Continuous variables are expressed as Mean ± Standard Deviation (SD). Inter-group comparisons of vitamin D levels were performed using one-way Analysis of Variance (ANOVA). Pearson's correlation coefficient was used to evaluate the linear relationship between serum vitamin D and thyroid parameters (TSH, FT3, FT4) within each patient subgroup. A p-value of < 0.05 was considered statistically significant for all tests.

RESULTS

Vitamin D levels varied across hypothyroid and euthyroid subjects as shown in [Table 1]. In

subclinical hypothyroidism, the mean vitamin D level was 20.65 ± 13.39 ng/mL. For overt hypothyroidism, the mean was 13.13 ± 12.14 ng/mL. Euthyroid individuals exhibited a mean vitamin D level of 48.73 ± 16.59 ng/mL.

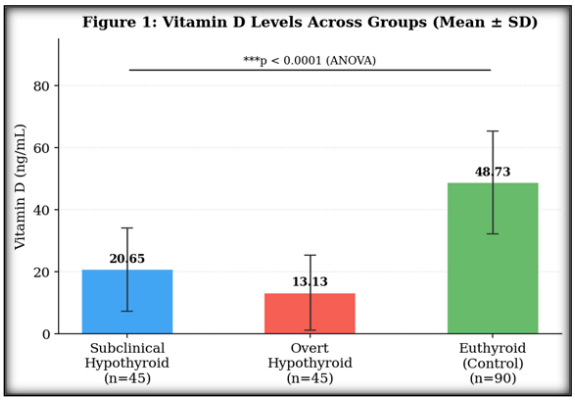
[Table 2] shows that both subclinical and overt hypothyroid patients had significantly lower vitamin D levels compared to euthyroid subjects ($p < 0.0001$, one-way ANOVA $F = 106.77$) [Figure 1].

Table 1: Status of Vitamin D Levels (Mean ± SD, ng/mL)

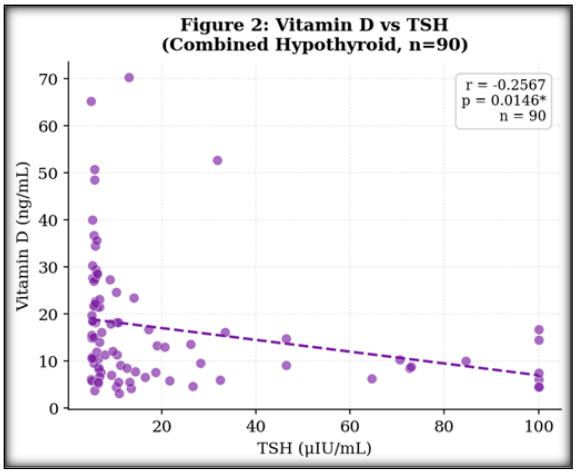
Group	Vitamin D (Mean ± SD, ng/mL)
Subclinical Hypothyroidism	20.65 ± 13.39
Overt Hypothyroidism	13.13 ± 12.14
Euthyroid (Control)	48.73 ± 16.59

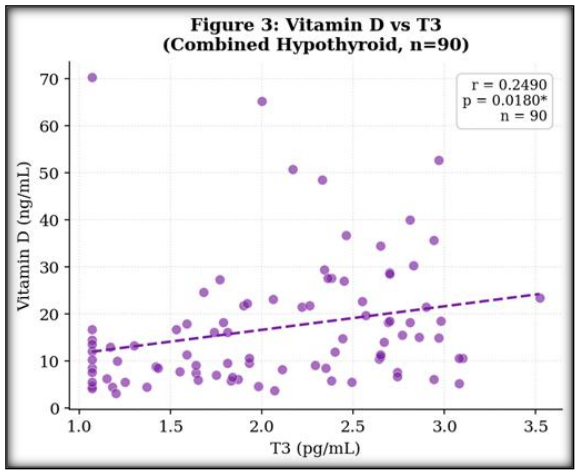
Table 2: Comparison of Vitamin D Levels Across Groups (ANOVA)

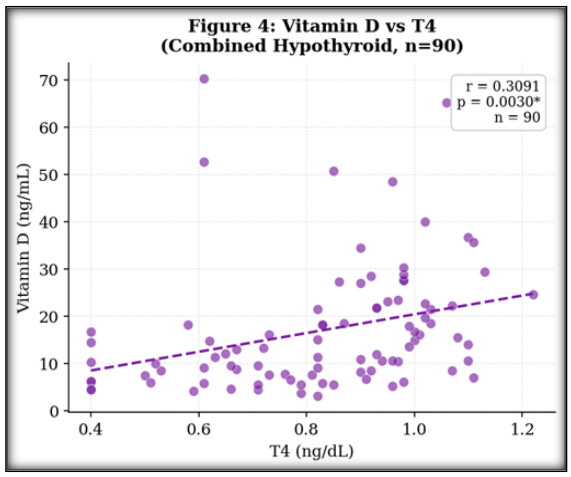
Group	Vitamin D (Mean ± SD)	F value	p value
Subclinical Hypothyroid	20.65 ± 13.39	106.77	< 0.0001
Overt Hypothyroid	13.13 ± 12.14		
Euthyroid	48.73 ± 16.59		



Pearson correlation analysis was performed on individual data from all hypothyroid subjects (n = 90) combined, as shown in [Table 3]. Vitamin D levels showed a statistically significant negative correlation with TSH ($r = -0.2567$, $p = 0.0146$) [Figure 2], a significant positive correlation with T3 ($r = 0.2490$, $p = 0.0180$) [Figure 3], and a significant positive correlation with T4 ($r = 0.3091$, $p = 0.0030$) [Figure 4].







Correlation of vitamin D levels with TSH, T3, and T4 in subclinical hypothyroid subjects (n = 45) was performed [Table 4]. The results revealed a negative correlation of vitamin D levels with TSH ($r = -0.2413$, $p = 0.1102$) [Figure 5]; however, the correlation was not found to be statistically significant. Vitamin D showed a negative correlation with T3 ($r = -0.1950$, $p = 0.1992$) [Figure 6], which was also not statistically significant. Vitamin D showed a statistically significant positive correlation with T4 ($r = 0.3015$, $p = 0.0442$) [Figure 7].

Table 3: Pearson Correlation of Vitamin D with TSH, T3, T4 in Combined Hypothyroid Subjects (n = 90)

	Vitamin D vs TSH	Vitamin D vs T3	Vitamin D vs T4
r value	−0.2567	0.2490	0.3091
p value (two-tailed)	0.0146*	0.0180*	0.0030*

* Statistically significant ($p < 0.05$)

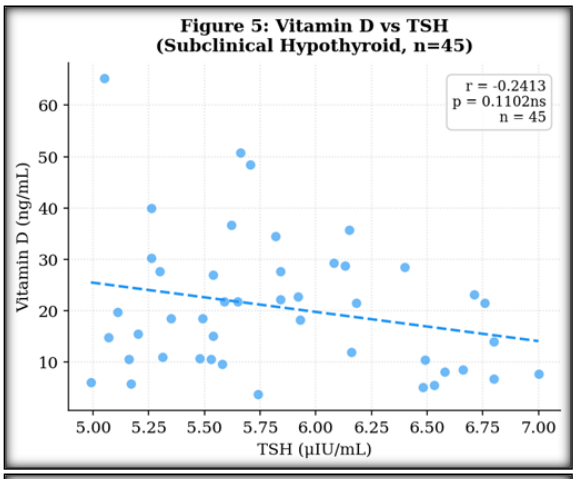
Table 4: Pearson Correlation of Vitamin D with TSH, T3, T4 in Subclinical Hypothyroid Patients (n = 45)

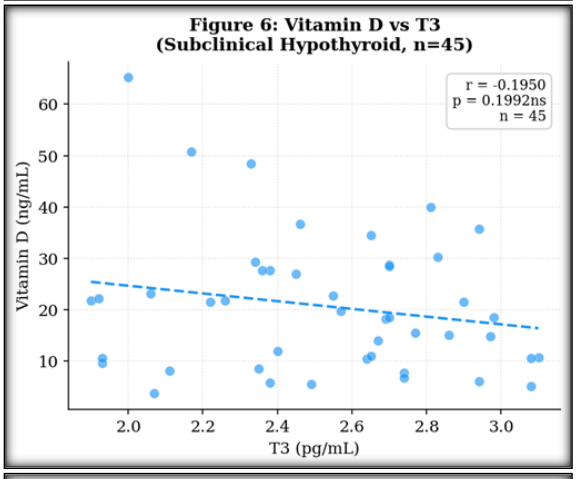
	Vitamin D vs TSH	Vitamin D vs T3	Vitamin D vs T4
r value	−0.2413	−0.1950	0.3015
p value (two-tailed)	0.1102	0.1992	0.0442*

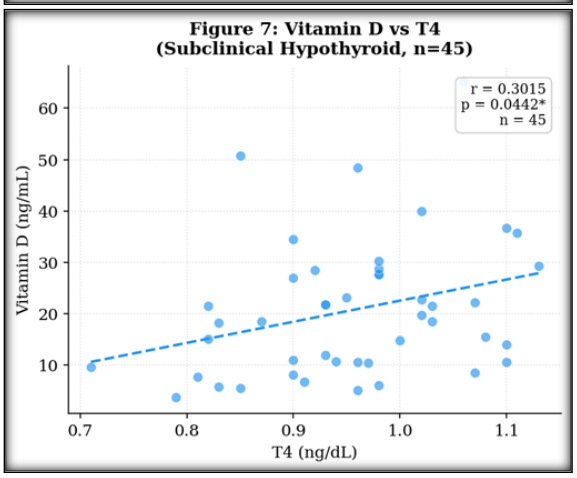
* Statistically significant ($p < 0.05$)

Table 5: Pearson Correlation of Vitamin D with TSH, T3, T4 in Overt Hypothyroid Patients (n = 45)

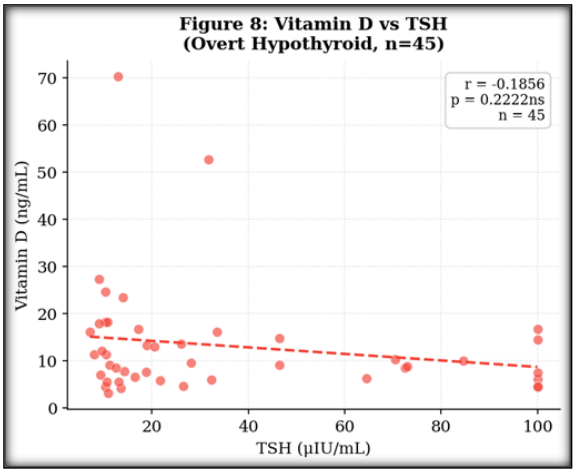
	Vitamin D vs TSH	Vitamin D vs T3	Vitamin D vs T4
r value	−0.1862	0.2547	0.1279
p value (two-tailed)	0.2208	0.0913	0.4025

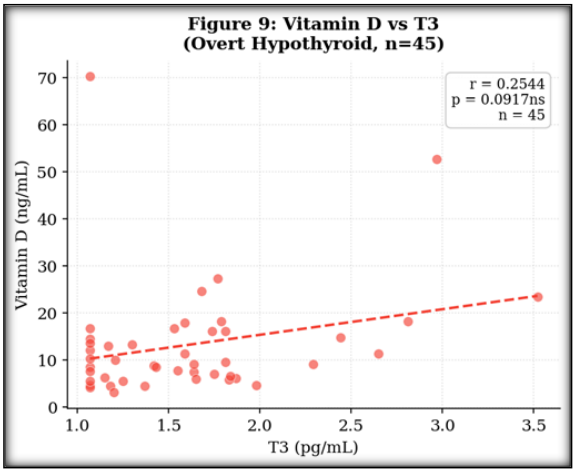


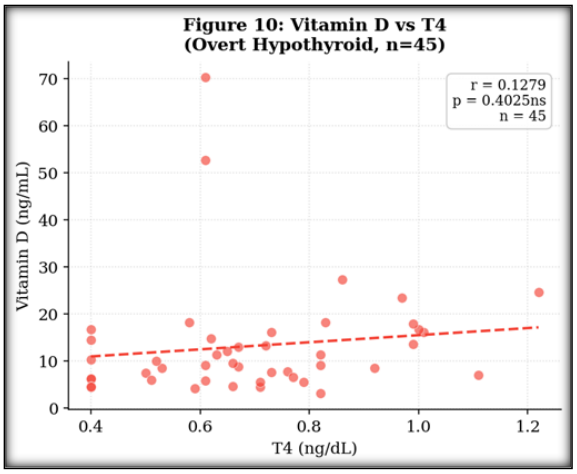




In overt hypothyroid patients (n = 45), vitamin D showed a negative correlation with TSH (r = −0.1862, p = 0.2208) [Figure 8]; however, the correlation was not found to be statistically significant. Vitamin D showed a positive correlation with T3 (r = 0.2547, p = 0.0913) [Figure 9] and T4 (r = 0.1279, p = 0.4025) [Figure 10], as shown in Table 5, and these correlations were also not statistically significant.







DISCUSSION

Hypothyroidism is the most common clinical indication of thyroid hormone deficiency.[6] The prevalence of hypothyroidism in Delhi is 13.2%.[5] Estimates suggest that up to 70–100% of the general population across the Indian subcontinent has insufficient vitamin D status, underscoring the scale of this public health problem.[3] Vitamin D deficiency can be a risk factor for hypothyroidism and autoimmune thyroid disease.[7]

Our study revealed that overt hypothyroid patients had significantly lower vitamin D levels (13.13 ± 12.14 ng/mL) compared to the euthyroid group (48.73 ± 16.59 ng/mL), and overt hypothyroid patients had significantly lower vitamin D than subclinical hypothyroid patients ($p < 0.0001$). The likely cause of low vitamin D levels could be insufficient intestinal absorption of vitamin D, or incomplete activation by the body.[8]

In a combined cohort of 90 hypothyroid subjects, Pearson correlation analysis on individual patient data revealed a statistically significant negative correlation of vitamin D with TSH ($r = −0.2567$, $p = 0.0146$), indicating that higher TSH (worse hypothyroidism) is associated with lower vitamin D. Vitamin D also showed significant positive correlations with T3 ($r = 0.2490$, $p = 0.0180$) and T4 ($r = 0.3091$, $p = 0.0030$), indicating that lower thyroid hormone levels are associated with lower vitamin D. The probable reason could be that similar steroid hormone receptors are bound by both thyroid hormone and vitamin D. Hypothyroidism may be linked to distinct gene expression through the vitamin D receptor.[2]

In subclinical hypothyroid patients (n = 45), vitamin D correlated negatively with TSH ($r = −0.2413$, $p = 0.1102$) and negatively with T3 ($r = −0.1950$, $p = 0.1992$), both non-significant. A statistically significant positive correlation was found with T4 ($r = 0.3015$, $p = 0.0442$). The probable reason may be that vitamin D deficiency, via nuclear receptor interactions, is linked to elevated TSH and reduced thyroid hormones.[9]

In overt hypothyroid patients (n = 45), vitamin D showed a negative correlation with TSH ($r = −0.1862$, $p = 0.2208$), which was not statistically significant. It has been demonstrated that vitamin D binds to a specific binding site in the pituitary and modulates TSH release; lower vitamin D may raise TSH levels.[10] Vitamin D showed a non-significant positive correlation with T3 ($r = 0.2547$, $p = 0.0913$) and T4 ($r = 0.1279$, $p = 0.4025$). The possible reason may be that 1,25(OH)2D3 binds to the nuclear high-affinity vitamin D receptor (VDR) found in most human tissues. When the activated VDR attaches to vitamin D response elements, gene expression is up- or down-regulated; thus low thyroid hormone levels may be associated with vitamin D deficiency.[11] More than 470 single nucleotide polymorphisms in the VDR gene modulate vitamin D's immunomodulatory impact; Apa1 and Fok1 are the two most common polymorphisms.[4]

CONCLUSION

Our study revealed the presence of vitamin D deficiency in both subclinical and overt hypothyroid patients compared to euthyroid controls. Pearson correlation analysis on individual patient data demonstrated a statistically significant negative correlation of vitamin D with TSH and significant positive correlations with T3 and T4. This shows that low vitamin D levels can exacerbate the systemic abnormalities linked to hypothyroidism. Supplementation with vitamin D and calcium may be recommended for patients with hypothyroidism. Routine screening for vitamin D deficiency in hypothyroid patients would enable timely intervention and reduce long-term morbidity.

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